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The Gaisbock syndrome after COVID-19 pneumonia

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ABSTRACT

In 1905, Felix Gaisbock, MD, described a syndrome in patients with hypertension, elevated hematocrit levels, plethoric appearances, and no splenomegaly. He postulated this relative erythrocytosis was due to stress. In this case report, a 40-year-old Caucasian man with obesity was admitted with recurrent deep vein thrombosis and increasing oxygen requirements 2 weeks after hospitalization with COVID-19 pneumonia. This patient had a 10-year history of untreated hypertension and erythrocytosis. He had a ruddy appearance, a normal erythropoietin level, and a negative JAK2 V617 mutation. In this case, Gaisbock syndrome was suspected.

KEYWORDS Erythrocytosis; Gaisbock syndrome; hypertension

In 1905, Felix Gaisbock, MD, described a syndrome with hypertension, erythrocytosis, and plethora without splenomegaly. He postulated this relative erythrocytosis was due to stress. The term "Gaisbock syndrome" originated.[1] This case report describes a 40-year-old man with a ruddy appearance found to have untreated hypertension over several medical encounters and incidental erythrocytosis spanning 10 years. This unusual syndrome still has clinical relevance.

CASE DESCRIPTION

A 40-year-old white obese (body mass index 46.5 kg/m^2) man was admitted with recurrent leg swelling and increasing oxygen requirements 2 weeks after hospitalization with COVID-19 pneumonia. He had untreated hypertension and erythrocytosis spanning 10 years *(Table 1)*. A deep vein thrombosis (DVT) in his leg diagnosed 2.5 months prior to his COVID infection had been treated with rivaroxaban. He reported daytime fatigue, frequent nighttime awakenings, and snoring but never had a sleep study or used continuous positive airway pressure. The patient reported a persistent rubor throughout his life. He had smoked 1 pack per day for 10 years but quit 12 years ago. He worked strenuous jobs in construction most of his life.

On this admission, the patient had a stocky, ruddy appearance; his blood pressure was 132/91 mm Hg, and his systolic blood pressures were intermittently high. Laboratory tests included a hemoglobin of 18.7 g/dL (13.7–17.5), a normal erythropoietin level of 5.7 MIU/mL (2.6–18.5), and negative JAK2 V617 mutation. He had a positive factor V Leiden mutation. He was not on any diuretics and did not appear volume depleted on the physical exam. He required a thrombectomy for persistent DVT with pain and swelling while on intravenous heparin and was discharged on rivaroxaban and oxygen at a flow rate of 6 L/min due to hypoxemia secondary to COVID-19. The patient had frequent anxiety attacks and was treated with alprazolam.

DISCUSSION

Erythrocytosis can be categorized as primary, secondary, or relative. Primary erythrocytosis, such as polycythemia vera, can be attributed to a JAK mutation, resulting in dysregulated production of erythrocytes, platelets, and myelocytes. Secondary erythrocytosis presents with an elevated erythropoietin level and an underlying cause of the erythrocytosis. Patients with relative erythrocytosis have a decreased plasma volume with a relative increase in hemoglobin, also known as hemoconcentration.

Evaluation of erythrocytosis should include a thorough history and physical examination. Tobacco use, medications (including androgenic steroids), carbon monoxide exposure, and signs of obstructive sleep apnea should be reviewed.[2] In

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Table 1. Hemoglobin levels and blood pressure trends of case subject over a 12-year period

Test	8/28/21	6/6/21	7/21/16	7/23/15	8/29/12	5/26/11	4/5/09
Hemoglobin (g/dL)	18.7	18.8	17.5	17.5	15	17.1	
Blood pressure (mm Hg)	132/91	175/102	137/90	128/94	133/84	137/85	155/83

this case, the patient's erythrocytosis was not consistent with primary or secondary erythrocytosis due to a negative JAK mutation and normal erythropoietin level. While hypoxemia secondary to COVID-19 pneumonia and sleep apnea could contribute to the patient's elevated hematocrit during this hospitalization, he had erythrocytosis for years prior to his COVID infection. He did not have an elevated erythropoietin level, as would be expected in hypoxemia and sleep apnea. Relative erythrocytosis, such as Gaisbock syndrome, is most likely given his history and laboratory tests.

Gaisbock syndrome is characterized by hypertension and erythrocytosis without splenomegaly, leukocytosis, or thrombocytosis. It is associated with mild obesity and increased blood viscosity, explaining why these patients often develop cardiovascular complications. These patients are also at a higher risk for thromboembolic complications.[3] Our patient presented with a recurrent DVT, ultimately requiring a thrombectomy. In the setting of Gaisbock syndrome, a positive factor V Leiden mutation, and recent COVID-19 infection, this patient is at especially high risk for thromboembolic events. Gaisbock attributed elevated hematocrit and hypertension to increased stress. He emphasized that these patients were often tense and anxious.[1] Hematocrit changes with hypertension have been observed in patients undergoing mental stress.[4]

Erythrocytosis has been associated with cardiovascular morbidity (odds ratio 1.8), cardiovascular mortality (hazard ratio 2.2), and all-cause mortality (hazard ratio 1.7).[5] A longitudinal study investigating cardiovascular events in adults reported that men between 65 and 95 years old with elevated hematocrit (49%–70%) had a higher risk of death due to congestive heart failure, coronary heart disease, intermittent claudication, and stroke.[6] In addition, documented cases have shown life-threatening thrombotic events, such as myocardial infarction and pulmonary embolism, in patients <40 years old with relative erythrocytosis.[7] This emphasizes the importance of recognizing erythrocytosis and investigating the underlying cause. Although first described in 1905, Gaisbock syndrome remains clinically relevant today.

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